

Cerebral oxygenation normalization is delayed following balloon atrial septostomy in neonates with transposition of the great arteries compared to preductal peripheral saturation.

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Introduction:

Fetal hypoxia has been implicated in the abnormal brain development seen in newborns with congenital heart disease. Following a balloon atrial septostomy (BAS) in patients with transposition of the great arteries (TGA) cerebral oxygenation increases rapidly but very little is known about the direct effect of BAS on the neonatal brain and on cerebral oxygenation. It is yet unclear how BAS affects cerebral oxygenation and if rapid increase of oxygen delivery results in brain reperfusion injury in neonates with TGA.

Methods:

We performed a prospective single center study to evaluate changes in the cerebral oxygenation before and for a period of 48 hours following BAS in 12 neonates with TGA. Data collected included peripheral oxygen saturation, mixed venous saturation, regional cerebral tissue oxygen saturation (r_cSO_2) measured by using near infrared spectroscopy, heart rate, blood pressure and hemoglobin prior and following BAS. Differences in the course of NIRS, preductal oxygen saturation, and calculated cerebral oxygen consumption between the two groups at different time points were analyzed. We also analyzed metabolic status of the patients including acidosis, lactate, and ischemia markers e.g. S100 before and following BAS.

Results:

9 (75%) patients underwent BAS at the age 4.6 (± 2.7) hours. Six (50 %) patients had prenatal diagnosis. There were 1 (1/12) early death prior to the septostomy. Lowest preductal peripheral oxygen saturation at admission was median of 64.5 (range 39.0 - 92.0), before BAS 85.6 (range 62.0 - 90.6), 5 minutes following BAS 86.8 (range 70.6 - 91.4) and 24 h after BAS 90.0 (range 85.2 - 93.6). Lowest (r_cSO_2) at same time points were 50.0 (range 35.0 - 70.0), 52.8 (range 36.4 - 72.5), 54.5 (range 37.2 - 73.8) and 69.2 (range 58.8 - 80.8).

Conclusions:

(r_cSO_2) levels rise at a slower rate than peripheral oxygen saturation values achieving stable level in 48 hours after BAS. Our data suggests that normalization of the cerebral oxygenation is delayed following the atrial septostomy. This may be due to brain reperfusion injury following the balloon atrial septostomy.

